Postural Vertigo and Positional Nystagmus

RUSSELL FLETCHER, M.D., *San Rafael*

• Oscillation of the eyes of a patient when the head is placed in a certain position is objective evidence to support a complaint of postural vertigo—dizziness when the head is tilted forward or upward or turned to one side or the other. Since positional nystagmus may be difficult to evoke and may be elicited at one time and not at another, it is important to make repeated tests, lest a causative lesion be overlooked.

Vertigo in such cases may be caused by pathologic change in the eighth peripheral nerve or in the central vestibular pathways. Sometimes no organic disease is observable even though positional nystagmus validates a complaint of vertigo. In such instances the patient should be assured that he does not have a progressive disease and be advised against activity in which dizziness would be hazardous.

MANY PATIENTS complain of dizziness when they change position, as in turning over in bed, stooping, looking overhead, or looking beneath a table. Usually the dizziness, called *postural vertigo,* is a momentary sensation. However, it may be pronounced and extremely disturbing and frightening. The attacks may last several seconds or minutes. Pronounced movement of the eyes, *positional nystagmus,* may be noted in many cases when the patient is examined during an episode of dizziness. Positional nystagmus may result from disease of the central vestibular tracts of the central nervous system, as well as from disease involving the peripheral or auditory mechanism.

Slight transitory attacks of postural vertigo are of no clinical significance, but thorough investigation is indicated if a patient complains of pronounced dizziness. In many cases if no pathologic change is noted in routine examination, the patient is told that there is nothing wrong with him. However, with careful *repeated* examination, positional nystagmus may be observed in a surprising number of such patients. And it is important to examine the patient carefully for the condition, for it is objective evidence of pronounced dizziness owing to disturbance of the vestibular mechanism by organic disease.

In positional nystagmus the movement of the eyes is so great that it can be seen at a distance of several feet. It is far greater than the nystagmus produced by the familiar caloric and Barany rotation tests and more active than the spontaneous nystagmus of albinos or that due to labyrinthitis. Unlike spontaneous nystagmus, which is present in the normal resting position and is not changed regardless of change of position, positional nystagmus occurs only when a certain position is assumed or upon a change of position. Positional nystagmus is not the same as that produced by the head-shaking test.

Tests for Positional Nystagmus. To observe a patient for positional nystagmus, attempt is made to put him in a position that will induce the dizziness of which he complains. In most cases this can be done simply by having the patient sit on a table and then go into the recumbent position and turn his head to the right or to the left. The motion may be carried out at varying rates of speed, but never with a violent jerk or twist of the head. Sometimes the nystagmus is latent—that is, it does not appear for five or ten seconds after the head is turned. Occasionally, hanging the head over the edge of the table may be necessary to elicit the movement, or patients who have dizziness when they stoop may be put in that position.

Frequently the patient will let the physician know when the dizziness starts and stops, and it can be observed that the nystagmus starts and stops simultaneously with the dizziness. The patient is often very apprehensive and may have pronounced sweating and pallor. Apparently the sensation is a frightening one, for many patients grasp the physician's arm at the onset of vertigo. Often patients close their eyes tightly, which makes it difficult to see the active nystagmus. However, it is possible to observe movements of the eyes beneath tightly closed lids. The nystagmus, which may last for from five seconds to a minute or more, is wild and active, and often it is difficult to determine whether the movement is horizontal, rotatory, or vertical.

It is extremely important that a patient who complains of postural vertigo be examined repeatedly, for quite often positional nystagmus will not be elicited the first two or three times the tests are car-

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ried out. This explains why one physician may report nystagmus (to an insurance company, for example), whereas another physician who has not elicited nystagmus may report no objective evidence of dizziness.

Pathologic factors and the mechanism by which positional nystagmus is produced were recently discussed in detail by Lindsay.[1]

There are three general classifications of cases of positional nystagmus. One comprises cases owing to disease involving the peripheral eighth nerve mechanism, which are of interest primarily to otologists. In a second group are cases of apparently spontaneous onset marked by repeated attacks off and on for several years, with no other objective findings of dizziness or of disease which might cause dizziness. It is probable that the condition derives from some vascular or vasomotor disturbance — most likely originating in the central nervous system, as the positional nystagmus is frequently of vertical type. Emotional factors may play a large part in such cases.

The third group comprises cases in which there is known to be severe head injury or other disease affecting the central nervous system, such as brain tumor or multiple sclerosis. In such cases a neurologist must determine the importance of positional nystagmus in relationship to other neurological findings. Positional nystagmus alone is not diagnostic of brain tumor or other disease of the central nervous system, but it may be one of the earliest signs of such a condition. It should be emphasized that positional nystagmus may be the only neurological sign of abnormality in a patient after convalescence from injury to the head and it may be the only objective evidence that the patient has incapacitating dizziness. In such circumstances the patient should be warned against work in which dizziness would be a hazard.

THERAPY

Except for those cases in which the dizziness is caused by peripheral disease of the ear or by disease of the central nervous system such as a brain tumor or head injury, there is no specific treatment. Use of Dramamine® and salt-free diet is ineffective. However, since many patients are greatly worried about the condition, it is important to reassure them, when no cause has been found, that they do not have a progressive degenerative disease and probably will gradually get well. Reassured, patients may accustom themselves to occasional dizziness so that they are not incapacitated.

1010 B Street.

REFERENCE

1. Lindsay, J. R.: Postural vertigo and positional nystagmus, Ann. Otol. Rhinol. Laryngol., p. 1135, Dec. 1951.

Discussion by* GILLIS A. ESSLINGER, M.D., *Berkeley

I agree with Dr. Fletcher that this condition is quite elusive. It is often as elusive as convulsive seizures so far as a physician's direct observation is concerned. Many times the physician is the last one to have the opportunity to give a personal account of an episode in a particular patient.

For this reason I also agree with Dr. Fletcher that frequent visits may be necessary and actual testing must be done. It seems to me according to my own experience that the objective evidence of the vertigo as indicated by the nystagmus may be more often elicited on the first visit and may not be as evident on subsequent visits. This may be evidence that increased emotional tone may favor the production of an episode through slight increase in irritability generally of the nervous system.

Through the work of Ernest Spiegel and other investigators it was found by a process of gradually eliminating different parts of the central nervous system that only a small portion of the nervous system is essential to the production of nystagmus. Specifically this includes the vestibular nucleus, the median longitudinal fasciculus, the nuclei of cranial nerves three, four and six and their peripheral extensions to their respective muscles.

Leidler about 1935 in reviewing over two hundred of his cases ascribed the severest vertigo to lesions of the eighth cranial nerve foot fibers and the adjoining brain stem.

De Kleyne about 1928 mentioned interference with the circulation of the vertebral artery in positions of extreme extension of the neck. This he felt was particularly true when the auricular artery was a direct branch from the vertebral artery. This may account for some of the cases of postural vertigo and positional nystagmus found in some types of cervical vertebra fracture and in platybasia.

Recent work of Groat and Simmons (1950) reveals when actual ganglion cell counts are done on guinea pigs suffering from concussion actual cell deficits are found. They examined some of their animals as late as thirteen months after injury. It was revealed that the greatest cell deficit from concussion occurred in the cells of the vestibular nucleus, reticular formation in the interior of the brain stem and the red nucleus. Cell deficits were found in every case of concussion. It was also of interest in this particular publication that the animals injured by concussion showed evidence of disturbance in behavior. Animals previously trained to run a maze appeared confused and unable to run the maze successfully without further training. It was also found that it was more difficult to train these animals to run a new maze and there was a tendency for them to forget their learning. It was found that this inefficiency persisted as long as thirty to ninety days.

It is felt that there may be a correlation in these bits of information with regard to a cause within the central nervous system in cases with the syndrome of this kind.

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Discussion by* VICTOR GOODHILL, M.D., *Los Angeles

Postural vertigo and positional nystagmus are symptoms of vestibular pathway hypersensitivity due to abnormal physiological stresses induced by postural changes. Dr. Fletcher has very clearly defined and illustrated these phenomena and the techniques by which they may be elicited. He has clearly indicated their variability and the necessity for repeated examination in some cases.

The elicitation of postural vertigo and positional nystagmus, however, should be considered as part of the investigation of labyrinth function. It should be preceded by routine otoscopic examination, audiometric studies and examination for spontaneous nystagmus, both overt and latent.

The phenomena of postural vertigo and positional nystagmus are *symptoms* of vestibular pathway dysfunction. They

are not clinical entities. The demonstration of postural vertigo and positional nystagmus in both peripheral and central disturbances of the vestibular pathway detracts from any localizing value in diagnosis.

The elaborate classifications of Nylen and others into "direction changing" and "direction fixed" types, have failed to yield localizing diagnostic data. The fact that repeated examination is necessary to elicit the response in many cases, speaks for a functional rather than an organic origin.

A curious fact has been mentioned by some investigators, recently Rucker of the Mayo Clinic. He has found the phenomenon more common in cases where the head is turned to the right in repose. He thus advises his patients to lie down with the head turned to the left. No explanation for this unilateral preponderance has been made.

In the elicitation of this phenomenon it is advisable to use plus 20 lenses in some cases to eliminate fixation and to amplify eyeball motions, as we frequently do in caloric testing.

In analyzing studies of vestibular dysfunction, it is helpful to think of:

a. Spontaneous nystagmus as evidence of constant hyperirritability and therefore organic.

b. Positional nystagmus as evidence of intermittent hyperirritability, and therefore probably functional.

c. Caloric—induced nystagmus — produced by abnormal stimulation.

d. Rotation—induced nystagmus — produced by hyperstimulation.

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Concluding Remarks of DR. FLETCHER

I wish to thank the discussants for their comments. Although I agree that positional nystagmus alone may not be diagnostic in localizing a lesion, this may be due to the fact that the medical profession has so far failed to find pathologic change to account for such nystagmus in all cases. I do not agree with the statement that "positional nystagmus is evidence of intermittent hyperirritability and is therefore probably functional." Epilepsy is intermittent irritability, but surely not functional. No normal person can produce positional nystagmus. A patient who has positional nystagmus following a severe head injury has objective evidence of severe dizziness which surely should not be classified as functional.
